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Attenuation of Renal Lipotoxicity via Restoration of Autophagy Flux through PI3K/AKT-GSK3 β -TFEB Signaling by Copper Oxide Nanoparticles

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Objectives : Obesity-related kidney disease is driven by renal lipid deposition, oxidative stress, inflammation, and impaired autophagy flux, which contribute to progressive renal injury. Therapeutic strategies targeting these pathogenic mechanisms remain limited. Copper oxide nanoparticles (CuO NPs), owing to their ultrasmall size and catalytic antioxidant capacity, have emerged as a potential therapeutic strategy, yet their mechanistic role in renal lipotoxicity remains unclear. In this study, we investigated the mechanistic effects of CuO NPs in renal lipotoxicity.

Methods : Ultrasmall CuO NPs (<4 nm) were synthesized via chemical reduction and applied to palmitate-treated HK-2 cells. Lipid accumulation, oxidative stress, fibrosis markers, and autophagy flux were assessed using Oil Red O staining, qPCR, immunoblotting, confocal microscopy, and pharmacological inhibitors targeting PI3K, AKT, and GSK3 β .

Results : CuO NPs significantly reduced palmitate-induced lipid accumulation and suppressed lipogenic genes (SREBF1, DGAT1, ACC). CuO NPs attenuated ROS production and downregulated MAPK activation. Fibrosis markers (Collagen IV, MMP2, α -SMA, Vimentin) were markedly decreased. CuO NPs restored impaired autophagy flux, evidenced by increased LC3B puncta under chloroquine treatment, and reduced p62 accumulation. Importantly, CuO NPs promoted TFEB nuclear translocation; these effects were abolished by PI3K or AKT inhibition and by GSK3 β constitutive activation, demonstrating that CuO NPs activate TFEB through PI3K/AKT–GSK3 β signaling.

Conclusions : CuO NPs exert potent antioxidant, anti-lipotoxic, and pro-autophagic effects in renal epithelial cells. By activating the PI3K/AKT–GSK3 β –TFEB axis, CuO NPs restore autophagy flux and mitigate lipid-driven oxidative and fibrotic injury. These findings support CuO NPs as a promising nanotherapeutic approach for obesity-related kidney disease.